mitral valve was noted in as many as 30 of 95 male patients with coronary heart disease. Systolic clicks were not heard in any of the cases studied. The finding of such a high percentage of mitral valve prolapse (MVP) in coronary heart disease is contrary to most published series on the systolic click-murmur syndrome (SCMS) where coronary angiography was invariably normal. The reason for this discrepancy lies in the fact that Dr. Aranda’s paper deals with papillary muscle dysfunction associated with coronary heart disease. On the other hand, the SCMS per se is a totally different entity in which the coronary vessels are usually normal and the MVP is subsequent to pathology involving the mitral valve apparatus or stems from left ventricular dysfunction.

We have followed up two patients with the SCMS and coronary heart disease demonstrated on angiography. Both were successfully operated (double and triple coronary bypass) with clinical and ergometric evidence of improved coronary supply. However, in both patients prolapse of the mitral valve persisted as did the atypical chest pains and palpitations associated with the SCMS. In view of this, and in view of our findings in the study of the SCMS before and after beta-blockade, we are of the opinion that in the SCMS the coronary arteries are invariably normal. In cases where both conditions coexist, we believe that we are dealing with two pathological entities rather than etiologically related conditions.

In the 30 male patients reported by the authors, the prolapse is part of the ischamic papillary muscle dysfunction complex and should be differentiated from the SCMS per se which is more common in females in whom typical anginal pains are lacking, coronary vessels are usually normal, systolic clicks are often present, the ST changes are not ischemic in nature, and in whom the myocardial lactate abnormalities were thought not to result from ischemia. Hence, one must differentiate between the SCMS and MVP secondary to ischemic papillary muscle dysfunction. The beta-blockade test may be of help in this context.

EDWARD G. ABINADER, M.B., MRCP I
Rothschild University Hospital
Haifa, Israel

References

The author replies:
To the Editor:

Prolapse of the scallops of the posterior leaflet of the mitral valve was detected angiographically in 30 of our 95 patients referred for evaluation of chest pain, arrhythmias or congestive heart failure. Most of these patients underwent saphenous vein aortocoronary bypass or aortoventriculotomy because their symptoms did not improve with medical therapy. Therefore the patients reported represent a selected group of patients with significant obstructive coronary artery disease (CAD) and dysfunction of the mitral valve apparatus. The prevalence of mitral valve prolapse (MVP) in a population with coronary artery disease remains to be determined.

Multiple conditions have been associated with the systolic click-midsystolic murmur syndrome, mitral valve prolapse syndrome or Barlow syndrome. The spectrum of clinical findings has varied from none to the presence of a nonejection click with or without a midsystolic murmur. As stated in Dr. Abinader’s letter, the MVP might be secondary to myxomatous changes in the valve, elongated or thickened chordae or left ventricular dysfunction. We believe that our group of patients represents a subset of patients within the clinical and pathologic spectrum which comprises the MVP syndrome. In this subgroup of patients, systolic clicks are rare. Impaired left ventricular contractility, hypertrophy and dilatation appear to be the factors responsible for MVP by causing distortion of the papillary muscle foundation during left ventricular contraction.

It is possible that in those cases where MVP and CAD coexist, one is dealing with double pathological entities rather than with etiologically related conditions as previously stated. Although pathological findings were not reported in our series, we subsequently had the opportunity to examine postmortem the hearts of two of the patients reported. The mitral valve and chordae tendineae were normal in the two specimens, but the papillary muscles were scarred with diffuse areas of fibrosis. Further clinical, angiographic and pathologic correlations are needed to elucidate this very important issue. Preliminary observations in our laboratory have shown that one can induce MVP in the dog after high grade stenosis of the left anterior descending and/or circumflex arteries.

We agree with the statement by Dr. Abinader that it is of utmost clinical importance to differentiate patients with MVP and normal coronary arteries from those with CAD and MVP. The following clinical profile may be useful in this respect:

| Sex: | Females predominate | Males predominate |
| Age: | 25-45 | >50 |
| Symptoms: | Asymptomatic or atypical chest pain and palpitations | Angina pectoris, congestive heart failure |
| Clinical findings: | Click or late systolic murmur is common | Click or late systolic murmur is rare |
| ECG: | Normal or non-specific changes | Abnormal >95% of cases, pathologic Q waves present in >50% |
| Beta blockade test: | Positive (?) | ? |
| Hemodynamic: | Normal resting intracardiac pressures | Elevated pressures, decreased CI and EF |
| Coronary arteries: | Normal | Extensive obstructive CAD |
| Treatment: | Inderal | Inderal or aortocoronary bypass surgery |
| Prognosis: | Benign | Guarded |
| Pathology: | Myxomatous degeneration of the valve, thickened chordae, normal papillary muscles | Infarction or fibrosis of papillary muscle, left ventricular scar and fibrosis, normal mitral valve leaflets |

JUAN M. ARANDA, M.D.
Veterans Administration Hospital
Miami, Florida 33125

References

BBB and H-V Prolongation

To the Editor:

We have read with great interest the article by Dhingra et al., which appeared in the April issue of Circulation. Although the findings and conclusions appear to contradict the observations made by Scheinman et al., there are several important differences in the groups of patients studied.

Twelve of the 18 patients reported by Dhingra had signs and symptoms of congestive heart failure, only one patient had primary conduction disease and two others developed neurological symptoms during the follow-up period. On the other hand, none of the
BBB and H-V prolongation.
J M Aranda, B Befeler and A Castellanos

Circulation. 1976;54:846-847
doi: 10.1161/01.CIR.54.5.846

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1976 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/54/5/846.citation